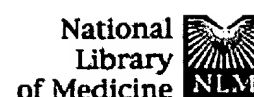


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☐ 1: Cell Signal. 1995 May;7(4):411-21.

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FULL-TEXT ARTICLE**Role of glycosyl-phosphatidylinositol hydrolysis as a mitogenic signal for epidermal growth factor.****Clemente R, Jones DR, Ochoa P, Romero G, Mato JM, Varela-Nieto I.**

Instituto de Investigaciones Biomedicas, Consejo Superior de Investigacione Cientificas (CSIC), Madrid, Spain.

We have investigated the role of the hydrolysis of glycosyl-phosphatidylinositol (GPI) as one of the signalling pathways elicited after interaction of epidermal growth factor (EGF) with its specific plasma membrane receptor (EGFR). Endogenous GPI was characterized in both NIH 3T3 mouse fibroblast cells and in EGFR-transfected NIH 3T3 cells (designated EGFR T17). GPI molecules isolated from both cell lines were identical and they incorporated radioactivity from both sugar and fatty acid substrates. Incubation of EGFR T17 cells with EGF, produced a rapid and transient hydrolysis of GPI. Maximum hydrolysis occurred after a 1-min incubation with 50 nM EGF. No such effects of EGF were observed in the parental cell line. Both inositol phosphoglycan (IPG)- and EGF-induced cell proliferation was inhibited in the presence of an IPG-antibody to different extents. The relationship between GPI hydrolysis and the activity of the EGFR was studied using the tyrosine kinase inhibitors tyrphostin (RG50864) and genistein. These agents were able to significantly inhibit EGF-mediated cell proliferation, EGF-dependent hydrolysis of GPI and EGF-regulated autophosphorylation of the EGFR. It is concluded that GPI hydrolysis is one of the earliest intracellular events generated in response to EGF.

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